IN THE UNITED STATES COURT OF FEDERAL CLAIMS
OFFICE OF SPECIAL MASTERS

No. 11-0654V
Filed: February 24, 2014
To Be Published

KEVIN RAYMO and HEATHER RAYMO, legal representatives of a minor child, HTR,	*	
Petitioners,	*	Tetanus Vaccine; Transverse Myelitis; TM; Autoimmune; Acute Transverse Myelitis; ATM; Disqualification of Experts; Factual Dispute Regarding Onset; Entitlement to Compensation.
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Jonathan Andry, Esq., Andry Law Group, LLC, New Orleans, LA for petitioners.
Claudia Gangi, Esq. and Ryan Pyles, Esq., U.S. Dept. of Justice, Washington, D.C., for respondent.

RULING ON ENTITLEMENT[1]

Vowell, Chief Special Master:

On October 11, 2011, Kevin and Heather Raymo ["Mr. Raymo," "Mrs. Raymo," or "petitioners"] timely filed a petition for compensation under the National Vaccine Injury Compensation Program, 42 U.S.C. §300aa-10, *et seq*.[2] [the "Vaccine Act" or "Program"], on behalf of their minor daughter, HTR. The petition alleges that HTR developed transverse myelitis[3] as the result of the human papillomavirus virus ["HPV"],

[1] Because this ruling contains a reasoned explanation for my action in this case it will be publically available, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (Dec. 17, 2002). Vaccine Rule 18(b) permits either party 14 days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Absent such a request, the entire ruling will be available to the public after 14 days have elapsed.

[2] National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755 (1986). Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa.

[3] According to the testimony in this case, the definition of the term "transverse myelitis" has evolved over the years. Tr. at 93-94, 118-19, 199. "Myelitis" refers to an inflammatory condition, but the term

meningococcal, hepatitis A, and diphtheria, tetanus and pertussis ["Tdap"][4] vaccinations she received on October 13, 2010. Petition at 1. For the reasons discussed below, I hold that petitioners have met their burden to show by preponderant evidence that a covered vaccine caused HTR's condition.

Few cases could be more heart wrenching than this one. Prior to her receipt of her sixth tetanus-containing vaccination[5] on October 13, 2010, HTR was a healthy eleven year old girl who liked art and played softball. Four days later, she was admitted to Arkansas Children's Hospital with loss of function in her legs. Her condition quickly progressed to complete paralysis below the T10 vertebrae in her spine with a complete loss of bowel and bladder control. In spite of rapid administration of therapies focused on a presumptive diagnosis of transverse myelitis, HTR was one of approximately one-third of those with that diagnosis who show no improvement with therapy.[6] She never regained any of her lost functions. Today, she is wheelchair-dependent, requiring frequent catheterization for urinary function and assistance from her parents for bowel function. Clearly, her life has been altered forever by what transpired after her October 2010 vaccinations.

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transverse myelitis is frequently used to refer to spinal cord injuries that do not have an inflammatory cause as well as to those that do. The more precise term for this broader category of spinal cord injury is "transverse myelopathy." *See* Transverse Myelitis Consortium Working Group, *Proposed diagnostic criteria and nosology of acute transverse* myelitis, NEUROLOGY, 59: 499-505 (2002), filed as Petitioners' Exhibit ["Pet. Ex."] 35 and Respondent's Exhibit ["Res. Ex."] D [hereinafter "TMCWG Diagnostic Criteria, Res. Ex. D"] at 499-500 ("Acute transverse myelopathy (which includes noninflammatory causes) and [acute transverse myelitis] have often been used interchangeably throughout the published literature. . . . As the clinical syndrome of acute transverse myelopathy may have noninflammatory cause (i.e. vascular causes), [acute transverse myelitis] represents a subset of acute myelopathy.") As used in this opinion, the abbreviation "TM" refers to the global and broad category of spinal cord injuries involving a loss of function below a transverse plane, regardless of cause. I use the abbreviation "ATM" to refer to the specific diagnosis of acute transverse myelitis. In their reports and testimony, the treating physicians and experts occasionally used the terms "transverse myelitis" and "transverse myelopathy" interchangeably, causing some confusion. *See, e.g.,* Tr. at 93-95, 117-19, 353-54; Res. Ex. J at 1, 5.

[4] Although HTR's medical records confirm that she received a Tdap vaccine on October 13, 2010 (Pet. Ex. 5, p. 1), the testimony and expert reports in this matter sometimes refer to the vaccination as her sixth DTaP vaccine. *E.g.* Tr. at 11-12, 55-56, 226-27; Pet. Ex. 9 at 1; Res. Ex. J at 1. The difference between DTaP and Tdap is not relevant to the issue of entitlement, other than to note that the Tdap vaccine contains a full dose of tetanus and reduced (booster) doses of pertussis and diphtheria. K. Stratton, et al., ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY (2012) [hereinafter "2012 IOM Report"] at 530 n.1.

[5] Although the petition claimed that all of the vaccines received on October 13, 2010, were causal, petitioners ultimately relied on a theory that implicated the tetanus component of the Tdap vaccination.

[6] More than one third of those with TM have a rapidly progressive course with a poor outcome (death or inability to ambulate). E. Frohman and D. Wingerchuk, *Transverse Myelitis*, NEW ENGL. J. MED., 363:564-72 (2010), filed as Pet. Exs. 11, 36 and Res. Ex. C [hereinafter "Frohman and Wingerchuk, Pet. Ex. 11"] at 565.

This case also presents several unusual features. Between filing the petition (a little less than a year after the events in question) and the causation hearing in Little Rock, Arkansas in November 2012, petitioners altered their causation theory several times. Although alteration of a causation theory is relatively common, petitioners proceeded to hearing on mutually exclusive causation theories based on different fact scenarios and different diagnoses (ATM or a spinal cord infarction). One of petitioners' experts, Dr. Daniel Becker[7] agreed with respondent's primary expert, Dr. John Sladky,[8] concerning both diagnosis and factual scenario, although they disagreed on the precise mechanism of injury. Unfortunately, both of these experts were discredited for reasons having little to do with their medical opinions and a great deal to do with their lack of candor.[9]

Another unusual feature in this case is that the filed medical records support both factual scenarios upon which the experts based their opinions because the records are somewhat vague and contradictory about when onset of HTR's symptoms occurred. Determining precisely when onset of HTR's first symptoms of transverse myelitis

[7] Doctor Becker received his medical degree from the Ruprecht-Karls University School of Medicine in Heidelberg, Germany. After two years as a neuroscience research assistant at the Washington University School of Medicine, Dr. Becker completed residencies in internal medicine and neurology at Vanderbilt University. He then completed two fellowships at John Hopkins University (spinal cord injury-medicine and intraoperative monitoring) and a fellowship at Duke University (transcranial magnetic stimulation). Doctor Becker is currently an Assistant Professor at Johns Hopkins University and Director of the Pediatric Spinal Cord Injury Unit at the Kennedy Krieger Institute. He is board certified in neurology, with a subspecialty certification in Spinal Cord Injury Medicine. He also holds a certificate as a Disability Analyst and is a Fellow of the American Board of Disability Analysts. He has around 25 publications, comprised of peer-reviewed articles, case reports, abstracts, and book chapters. *See generally* Tr. at 86-95; Pet. Ex. 33.

[8] Doctor Sladky obtained his undergraduate and medical degrees from Yale University. He completed his residency in pediatrics at the Yale-New Haven Hospital and a fellowship in neurology at the Children's Hospital of Philadelphia ["CHOP"]. Doctor Sladky also was an MDA clinical neuromuscular Fellow at CHOP and an MDA research Fellow at the Hospital of the University of Pennsylvania's Department of Neurology. Upon completion of his fellowships in 1984, he was appointed an Assistant Professor in both the pediatrics and neurology departments at the University of Pennsylvania and also held clinical positions at CHOP. In 1995, three years after being named an Associate Professor, Dr. Sladky left the University of Pennsylvania to accept a position with Emory University. He remained affiliated with Emory for approximately fifteen years before leaving to join a private clinical practice. Doctor Sladky is board certified in pediatrics, neurology (with a special competence in child neurology), and electrodiagnostic medicine. He has published original research papers, review articles, and abstracts. *See generally* Tr. at 187-91; Res. Ex. B.

[9] Petitioners appear to have acknowledged, albeit implicitly, the credibility and reliability problems posed by Dr. Becker's report and testimony in that petitioners do not rely upon Dr. Becker's theory of causation in their post hearing brief. *See* Petitioners' Post Hearing Brief ["Pet. Post Hearing Brief"] at 9-10 (arguing "[HTR's] paralysis as the result of an autoimmune condition as opposed to an infarct in her spinal cord"). Likewise, respondent brought Dr. Sladky's credibility problems to the court's attention in a supplemental post hearing filing. *See* Respondent's Status Report, filed May 1, 2013 ["May Status Report"], at 1-2. However, respondent had no theory other than Dr. Sladky's upon which to rely, and no other witness to counter Dr. Kinsbourne's theory that HTR suffered from ATM.

occurred is crucial to the causation opinions. If HTR's symptoms progressed from onset to nadir (the point of maximum impairment) in less than four hours, the autoimmune causation theory lacks a factual basis, according to Dr. Marcel Kinsbourne,[10] the expert who advanced the theory. Under these circumstances, I find the Mrs. Raymo's affidavit and her expanded but consistent testimony to be the deciding factors in determining when onset of HTR's symptoms occurred.

I. Procedural History.

In addition to filing their petition on October 11, 2011, petitioners filed an expert report from Dr. Kinsbourne, Mrs. Raymo's affidavit, and six exhibits containing medical records. *See* Pet. Exs. 1-8. Petitioners filed Dr. Kinsbourne's supplemental expert[11] with supporting medical literature on December 13, 2011. *See* Pet. Exs. 9-24.

On February 8, 2012, respondent filed her Rule 4(c) report. She also filed the expert report and curriculum vitae ["CV"] of Dr. John Sladky and six supporting medical literature articles. *See* Res. Exs. A-H. While Dr. Kinsbourne had described HTR's injury as ATM (Pet. Ex. 9 at 2-3), Dr. Sladky classified it as a spinal cord infarction ["SCI"], likely caused by a fibro-cartilaginous embolism ["FCE"] (Res. Ex. A at 6). After reviewing respondent's Rule 4(c) report and the report of Dr. Sladky, Dr. Kinsbourne recommended that petitioners consult a neuroradiologist to review HTR's MRI images and determine if the alternative diagnosis proposed by Dr. Sladky could be correct. *See* Petitioners' Response to Respondent's Rule 4(c) Report ["Rule 4(c) Response"], filed April 30, 2012, at 9.

[10] Doctor Kinsbourne received his medical education and training at Oxford University. After earning his medical degree in 1955, he spent nine years obtaining specialty training in the disciplines of pediatrics, neurology, child neurology, and neurosurgery. In 1964, he became a lecturer in the university's cognitive neuroscience department. He moved to the United States three years later to be a professor and chair of the child neurology division within the pediatrics department at Duke University. Although he had obtained the British equivalents, Duke requested that Dr. Kinsbourne sit for an American medical board examination. He sat and passed the examination in pediatrics. After seven years at Duke University, Dr. Kinsbourne moved to a position at the University of Toronto and Hospital for Sick Children. In 1980, he transitioned to a more research-orientated position at the Eunice Shriver Kennedy Center in Massachusetts. For approximately the past twenty years, Dr. Kinsbourne has taught clinical psychology to graduate students at the New School in New York. Over the course of his career, he has published over 400 articles, consisting of peer-reviewed articles, textbook chapters, and theoretical articles. Additionally, Dr. Kinsbourne has written or edited ten monographs and books. *See generally* Tr. at 26-31.

[11] I requested the supplemental opinion of Dr. Kinsbourne because he relied upon the conclusions regarding a relationship between the tetanus vaccine and TM contained in K. Stratton, et al., ADVERSE EVENTS ASSOCIATED WITH CHILDHOOD VACCINES: EVIDENCE BEARING ON CAUSALITY (1994) [hereinafter "1994 IOM Report"]. *See* Order, issued Nov. 4, 2011, at 1. A more recent report from the Institute of Medicine, released on August 25, 2011, and published in 2012, also addressed the evidence regarding a relationship and I wanted the benefit of Dr. Kinsbourne's views on the more recent report.

Petitioners took Dr. Kinsbourne's recommendation, and filed expert reports from Dr. Patrick Barnes,[12] a pediatric neuroradiologist, and Dr. Becker, a neurologist. *See* Pet. Exs. 29, 31-32. Petitioners also attempted to obtain an expert report from Dr. Cheran Shah, the treating physician who initially reviewed the MRI at Arkansas Children's Hospital. After reading Dr. Sladky's report and re-reviewing the MRI images, Dr. Shah indicated to them that he remained confident in his initial diagnosis of ATM. According to petitioners, hospital policy prevented Dr. Shah from preparing a written statement or otherwise being involved in this case. Rule 4(c) Response at 3-4.

Based on his review of HTR's MRI, Dr. Barnes concluded that the "spinal cord findings [were] not consistent with infarctions or ischemic myelopathy (*e.g*. embolic, thrombotic, or hypoperfusion) unless associated with an infectious or post-infectious vasculopathy, vasculitis, or hypercoagulopathy." Pet. Ex. 29 at 2. He did not explain why the cause of an infarction would affect the spinal cord's appearance on the MRI. Doctor Becker agreed with Dr. Sladky's classification of HTR's injury as an SCI, but he opined that the infarction resulted from vascular thrombosis trigged by her HPV vaccination rather than from an FCE. Pet. Ex. 31 at 3. Thus, Dr. Becker's opinion dovetailed with that of Dr. Barnes.

On July 2, 2012 respondent filed a supplemental Rule 4(c) report, a supplemental expert report from Dr. Sladky, and an expert report and CV from pediatric hematologist Dr. Joan Cox Gill.[13] *See* Res. Exs. I-K. Both reports addressed the HPV-based causation theory put forth in Dr. Becker's expert report.

The parties filed their pre-hearing submissions on November 6, 2012. Although petitioners disavowed Dr. Kinsbourne's causation theory in their April 2012 Rule 4(c)

[12] Doctor Barnes attended the University of Oklahoma for both his undergraduate and medical studies. He completed his residency in diagnostic radiology in Oklahoma. He completed a fellowship in pediatric neuroradiology and cardiovascular radiology at Harvard Medical School and the Children's Hospital in Boston, MA, in 1977, Doctor Barnes is board certified in diagnostic radiology, with an added certification in neuroradiology. He currently is the Chief of the Pediatric Neuroradiology Section and Co-Director of the Pediatric MRI and CT Center at Lucile Salter Packard Children's Hospital and Stanford University Medical Center. *See generally* Pet. Exs. 29 (expert report) and 30 (CV).

[13] Doctor Gill received a B.S. from St. Norbert College and her medical degree from the Medical College of Wisconsin. She completed her pediatric internship and residency at the Milwaukee Children's Hospital and did a fellowship in Pediatric Hematology-Oncology through the Medical College of Wisconsin and the Blood Center of Southeastern Wisconsin. Since completing her fellowship in 1981, Dr. Gill has held a faculty appointment with the Medical College of Wisconsin. She is currently a Professor of Pediatrics, Medicine, and Epidemiology. Additionally, Dr. Gill is the Director of the Comprehensive Center for Bleeding Disorders at The Blood Center of Wisconsin and the Medical Director of the Hemophilia and Bleeding Disorders Center at the Children's Hospital of Wisconsin. Doctor Gill serves on several medical and professional committees and is board certified in both pediatrics and pediatric hematology/oncology. *See generally* Tr. at 302-11; Res. Ex. K.

Response,[14] the pre-hearing submissions indicated that they would be presenting evidence on both a Tdap-ATM causation theory and an HPV-SCI theory. Petitioners' Memorandum for Entitlement Hearing at 4-5; Parties' Joint Prehearing Submission at 2.

Mrs. Raymo and Drs. Kinsbourne, Becker, Sladky and Gill testified in person at the entitlement hearing held in Little Rock, AR on November 27 and 28, 2012. At the conclusion of the hearing, petitioners requested the opportunity to file post hearing briefs. Petitioners filed their post hearing brief on February 6, 2013. Respondent's responsive brief was filed on February 22, 2013.

After the hearing, two issues arose which are discussed in detail in Section IV: Dr. Becker's apparent plagarization of another expert's report and Dr. Sladky's failure to disclose the suspension of his medical license to respondent. I provided both parties an opportunity to address these issues prior to issuing this decision. This case is now ripe for a ruling on the issue of entitlement.

II. Relevant Medical History and Factual Findings.

A. Undisputed Facts.

HTR was born at Keesler Air Force Base on February 2, 1999. Pet. Ex. 3, p. 1. Her early childhood growth and development were normal. *See generally* Pet. Exs. 4, 8. She had recurrent ear infections and allergies, but no serious health concerns during her first eleven years of life. *Id.* HTR received recommended childhood vaccines with no reported ill effects. *See* Pet. Exs. 4, 5.

On Wednesday, October 13, 2010, HTR was seen for a headache and sinus drainage. She also complained of congestion and cough, which had persisted for two weeks. Pet. Ex. 4, p. 148. HTR was diagnosed with allergic rhinitis and an upper respiratory infection, for which she was prescribed Claritin and Sudafed. Pet. Ex. 4, p. 149; Tr. at 11.

HTR also received the allegedly causal vaccines on October 13, 2010. After the visit for her allergies, HTR went to the immunization clinic where she was administered her second hepatitis A and first HPV (Gardasil), meningococcal, and Tdap vaccinations.

[14] In their response, petitioners quoted respondent's Rule 4(c) conclusion that they had "not provided a reputable medical theory causally connecting [HTR's] Tdap vaccination with her paraplegia and related complications" before stating that they "do not dispute that the expert report of Dr. Kinsbourne is not a reputable medical theory in this matter. Dr. Kinsbourne based his entire analysis and resulting opinion upon the medical opinions of the treating physicians that [HTR] developed [A]TM . . . it was not unreasonable for [HTR's] treating physicians and Dr. Kinsbourne to believe from a review of the MRI images that the diagnosis of [A]TM to be correct. However, it now appears likely to Petitioners, after reviewing the reports of Dr. Sladky and Dr. Becker, that the original diagnosis of [A]TM by the treating physician is incorrect." Rule 4(c) Response at 10-11.

Pet. Ex. 5, p. 151; Tr. at 11-12. No time-stamped records were filed from the immunization clinic, but per Mrs. Raymo's testimony, HTR received the vaccinations around 11:30 AM.[15]

On Thursday, October 14 and Friday, October 15, 2010, HTR went to school. She did not complain of any ill effects from the vaccinations. Tr. at 13-14. Friday night she attended an overnight lock-in program at her church. When picked up Saturday morning, HTR was a bit grumpy from a lack of sleep, but otherwise seemed like her typical, healthy self. Tr. at 14-15. Her paternal grandparents visited the family that weekend, and she spent Saturday walking around and sightseeing with them. *Id.* That night, with HTR's grandparents staying in the master bedroom, Mrs. Raymo slept in HTR's room and Mr. Raymo slept in HTR's sister's room. HTR went to bed around 9:00 PM and to sleep around 9:30. She was still asleep when her mother woke up around 7:30 AM on Sunday, October 17, 2010. Tr. at 16-17.

By the afternoon of October 17, 2010, HTR was admitted to the hospital with a presumptive diagnosis of TM. She was completely paralyzed from the waist down. Pet. Ex. 7, p. 195.

B. Disputed Facts.

1. Overview.

The parties' most significant disagreement, other than that of causation itself, involves timing. Three periods are at issue: (1) when HTR first experienced symptoms of TM; (2) the length of time between the first symptom and the nadir of her symptoms; and (3) the time between vaccination and onset of symptoms. Resolution of these factual disagreements is critical to analyzing petitioners' causation theory because Dr. Kinsbourne unequivocally testified that he could not support an autoimmune theory if onset to nadir was under four hours. Tr. at 37-38.

2. Law Pertinent to Factual Conflicts.

Conflicts between contemporaneous records and testimony given several years later at a hearing are common in Vaccine Act cases, and this case is no exception. Two general legal principles guide the resolution of conflicts between contemporaneous records and later-adduced evidence. The first is that the absence of a reference to specific symptoms in a medical record does not conclusively establish the absence of symptoms during that time frame. *See, e.g.*, *Murphy v. Sec'y, HHS*, 23 Cl. Ct. 726, 733 (1991), *aff'd*, 968 F.2d 1226 (Fed. Cir. 1992) ("[T]he absence of a reference to a

[15] Tr. at 12. The notes from HTR's morning appointment indicate it began around 10:30 AM and that the visit was concluded by 10:59. Pet. Ex. 4, pp. 148-49.

condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance") (citation omitted).

The second principle addresses the degree of reliance commonly accorded to contemporaneous records. Special masters frequently accord more weight to contemporaneously-recorded medical symptoms than those recounted in later medical histories, affidavits, or trial testimony. "It has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight." *Murphy*, 23 Cl. Ct. at 733 (citation omitted); *see also Cucuras v. Sec'y*, *HHS*, 993 F.2d 1525, 1528 (Fed. Cir. 1993) (medical records are generally trustworthy evidence). Memories are generally better the closer in time to the occurrence reported and when the motivation for accurate explication of symptoms is more immediate. *Reusser v. Sec'y, HHS*, 28 Fed. Cl. 516, 523 (1993). Inconsistencies between testimony and contemporaneous records may be overcome by "clear, cogent, and consistent testimony" explaining the discrepancies. *Stevens v. Sec'y, HHS*, No. 90-221V, 1990 WL 608693, at *3 (Fed. Cl. Spec. Mstr. Dec. 21, 1990).

3. Nature of the Dispute.

The parties agree that at some point during the morning hours of October 18, 2010, HTR began to experience numbness in her right leg, that the symptoms spread to her other leg, and resulted in paralysis of HTR's lower body by the time of her neurological examination at the second hospital she visited that morning. Petitioners contend that HTR first noticed the symptoms of a transverse myelopathy upon awakening and that onset therefore occurred the prior evening.[16] If petitioners are correct, symptom onset likely occurred while HTR was sleeping. Respondent takes the position that HTR was entirely normal when she awoke, and only began experiencing symptoms of a transverse myelopathy when she started to get out of bed.

Based on their differing positions as to the timing of onset of HTR's symptoms, the parties also disagree on the length of time between onset and nadir. Petitioners contend that it took longer than four hours, while respondent contends nadir was reached within four hours.

4. Evidence.

The medical records discussed below, particularly those from the emergency room at Baptist Health Medical Center ["BHMC"] and from HTR's transfer and

[16] Petitioners rely upon a late-filed medical article (Pet. Ex. 57) to place onset of symptoms at the time HTR went to sleep. Pet. Post Hearing Brief at 5 (citing H. Adams, *et al.*, *AHA/ASA Guideline: Guidelines for the Early Management of Adults with Ischemic Stroke*, STROKE, 38:1655-1711 (2007)). Respondent objected to my consideration of this article. *See* Respondent's Post Hearing Brief ["Res. Post Hearing Brief"] at 5. Although I examined the document, I place no reliance on it. The document concerns a medical "rule of thumb" for administration of "clot busting drugs" in patients who awaken with symptoms of a brain stroke. This presumption is not relevant to my factual determination.

admission to Arkansas Children's Hospital ["ACH"], are somewhat inconsistent in identifying a time of onset. These inconsistencies reflect a lack of clarity in the questions asked and the answers given. Given HTR's condition, determining precisely when the first symptoms arose was unnecessary to determine a course of treatment.[17] Unlike a brain stroke,[18] where careful questioning regarding symptom onset is necessary to determine if "clot-busting" drugs may be administered, medical personnel treating HTR were focused on determining what was wrong, and Mrs. Raymo and HTR's responses likely reflected the near hysteria experienced over HTR's rapidly progressing paralysis.

a. The Contemporaneous Medical Records.

(1) Records from Baptist Health Medical Center.

HTR arrived at BHMC at 9:18 AM on the morning of October 17, 2010. She was evaluated by an emergency room triage nurse at 9:35 AM. Pet. Ex. 6, p. 157. The nurse recorded a chief complaint of "[right] flank pain then tried to stand up and was unable to walk." *Id.* The triage report reflects paresthesia, decreased range of motion, and weakness in HTR's legs. The leg weakness and numbness were worse in the right leg than the left. *Id.*, p. 158.

The typed Emergency Department History and Physical note, dictated by Dr. Chris Hall, provides additional details about what occurred prior to their arrival at BHMC:

> Mother says [HTR] has complained of some right flank pain, and when she tried to stand up she was unable to walk. She said her right leg felt numb and tingling. She had weakness in her right leg and some paresthesias and weakness in the left, but mainly in the right. The patient has had a cough and congestion 2 to 3 days ago, but has otherwise not

[17] TM symptoms are treated as if they are inflammatory or autoimmune in origin. Corticosteroids are the generally accepted first line treatment, although there have been no randomized controlled trials involving their use. Their use has been derived from case studies and extrapolation from patients with multiple sclerosis. If corticosteroids are ineffective, plasma exchange is usually the next alternative. Frohman and Wingerchuk, Pet. Ex. 11, at 568-69. However, there are no professional guidelines for treatment and management. *Id*. at 571. HTR did not respond to either treatment, placing her among the approximately one-third of those with TM who have a poor outcome in spite of treatment. TMCWG Diagnostic Criteria, Pet. Ex. D, at 499. The rapid progression of symptoms and spinal shock were risk factors predictive of a poor likelihood of recovery. *Id.*

[18] Timing can be important in assigning a possible or probable cause for TM, but it has little bearing on treatment. A person presenting with symptoms of TM would be treated as if the condition were immune-mediated, as there is no treatment for a spinal cord infarction. *See* TMCWG Diagnostic Criteria at 501 (noting that some vascular myelopathies may fall within the ATM guidelines and recommending that, patients with suspected ATM should receive immediate treatment instead of waiting for nadir of symptoms); *see also* Tr. at 355-56 (clot-busting drugs are typically not used in the treatment of spinal cord infarctions because the act of administering them could cause an even greater harm).

> been sick and has not been running a fever. She complains of some right suprapubic abdominal pain and right flank pain.

Pet. Ex. 6, p. 154. Doctor Hall also indicated that:

> [HTR] either is unable or unwilling to move her right leg. Initially it seemed it could be somewhat voluntary, but I could not get her to move it at all and in fact, when she was taken to the bathroom to get a urine specimen her mother had to lift her back into the chair. She was unable to support her weight and basically slumped to the floor. She would not even bear weight with her left. I could not get her to show any strength in the right leg. It was 0 out of 5 strength. It was somewhat flaccid. Her left leg she (sic) would not do any strength at all. Initially, it seemed that she was moving it somewhat, but on re-exam there is still very little movement in the left leg, if any at all. She just would say she cannot move it. She did have some sensation in the left leg to pinprick in the lower leg and upper leg, but it was somewhat dulled. In the left foot she had minimal to no sensation. In the right foot, right lower leg, and right upper leg, even to sharp pinprick she had no apparent sensation. She tells me that she can feel things somewhat but not as it should be, some sort of paresthesia, according to the patient.

Id., p. 155. The treating physicians decided to transfer HTR to ACH "for the purpose of specialized pediatric services not available at [BHMC]." Pet. Ex. 6, pp. 164 (Authorization/Consent For Transfer), 165 (Transfer Certification Statement). The records reflect the time of discharge as 12:03 PM. Pet. Ex. 6, pp. 160, 172.

(2) Records from ACH.

The medical records reflect the time of HTR's arrival at the ACH emergency room as 12:25 PM. Pet. Ex. 7, p. 347. Soon after her arrival, HTR was evaluated by neurologist Dr. William Walters. He summarized her presentation as:

> 11 yr old WF with [no prior medical history] of note who awoke with numbness right leg, also paralyzed followed < 1 hr by left leg, now with complete numbness & paralysis to belly button. Also c/o stomach tenderness; [no] recent fever or illness.

Id., p. 178. His assessment was that HTR was an "[a]cute onset paraplegic ~T10, probably transverse myelitis; considering stroke, virus, AIDP, polio like illness. MS, central lesion less likely." *Id.*, p. 179.

The records from ACH include a daily note for each day HTR was hospitalized. *See*, *e.g.*, Pet. Ex. 7, pp. 206-07 (Day 3 note), 217-18 (Day 5 note). These day notes were written contemporaneously, but more remotely from the events when HTR awoke

on October 17, 2010. The note for October 17, 2010, day 0, which was written at 11:31 PM, recaps the events of the day:

> Mom said that [HTR] got up this morning like she normally did and had no problems. A little while after she got up, she couldn't stand on her legs and fell down. She said that it felt like her legs were asleep. Mom took her to Springhill and she was still able to move her toes on her left foot but couldn't move her right leg at all. She could no longer feel her legs at all. Mom said that it started in her right leg and then moved to her left leg. She was also unable to urinate. Now she is unable to move either of her legs and is numb up to her hips. She has had a cough and runny nose for past week. In ER, they did several blood tests and she got an MRI of her spine which showed an area of increased uptake in the lower thoracic area, but is likely an artifact because she was moving. Dr. Walters has seen and examined the [patient] in the ER.

Pet. Ex. 7, p. 195.

On October 19, 2013, an infectious disease specialist, met with HTR and her mother. The notes from the meeting indicate that:

> According to pt's mom, she was seen by PCP on Wednesday 10/13 [secondary] to cough & "sinus infection." Pt was also given her Gardasil, menactra and tetanus vaccinations at that visit. Mom states that [HTR] had been taking Zyrtec intermittently for allergies, but [otherwise] was not taking any other medications. Mom states that pt continued to have mild cough and runny nose after the PCP visit but [no] other symptoms were present until yesterday 10/17. On yest., [HTR] states that she woke up and felt fine. A few minutes after awakening she began to have weakness and tingling in her R LE & when she tried to stand up she fell down. The pt taken to an [*sic*] OSH & while there she developed weakness & loss of sensation in her L LE also. The patient was subsequently transferred to ACH & upon arrival had lost all bowel and bladder control. Mother denies fever, headache, [nausea/vomiting], diarrhea, recent travel, sick contacts or tick exposure.

Pet. Ex. 7, p. 187.

b. Mrs. Raymo's Affidavit.

In her October 6, 2011 affidavit, Mrs. Raymo indicated that "[o]n the morning of October 17, 2010, when [HTR] awoke, she complained of weakness and tingling in her right leg. When she attempted to stand up, she fell down." Pet. Ex. 2 at ¶ 9. Upon arrival at BHMC, "[HTR] complained that her right leg was numb and [she] was experiencing weakness in the left leg." *Id.* at ¶ 11.

Mrs. Ramyo also reported that "[w]hile at [BHMC] [HTR] also developed loss of sensation in her left leg." Pet. Ex. 2 at ¶ 12. Approximately two and half hours later, the ACH "attending physician evaluated [HTR]. [HTR] had lost sensation in both of her legs to her belly button and was experiencing tenderness in her stomach and lower back. [HTR] had also lost bladder and bowel control." *Id.* at ¶ 15.

c. Mrs. Raymo's Testimony.

Mrs. Raymo's testimony provided further details about the events of October 16-17, 2010. Because Mr. Raymo's parents were staying in the master bedroom, Mrs. Raymo slept in HTR's room the night of October 16, 2010. Mrs. Raymo awoke around 7:30 AM and left the room to get dressed for church. Tr. at 16-17. When she returned to the room, HTR was awake and sitting "half on and half off the bed." Tr. at 17. HTR indicated that she couldn't feel her right leg. Mrs. Raymo thought the leg was just "asleep" and left the room to finish getting dressed. *Id.* When Mrs. Raymo returned to the room a second time, HTR stressed that she was not playing and that she could not feel her leg. Mrs. Raymo indicated she then tried pinching the leg and HTR could not feel it, which is when she realized something was wrong. *Id*.

Mrs. Raymo called her husband and they tried to get HTR to stand up, but she was not able to do so. Tr. at 17. HTR said that she could not feel her right side at all and that her left side was tingling and felt a little bit numb. Tr. at 17-18. She did not feel any pain in her legs or right side of her body. Tr. at 21. Mrs. Raymo asked HTR if she had noticed anything being different during the night. HTR replied that she had not. Tr. at 18.

Rather than waiting for an ambulance to arrive, Mr. and Mrs. Raymo put HTR into a computer chair and then transferred her into their car. Tr. at 17. They arrived at BHMC by 10:00 AM. Tr. at 18. They stayed there for about two hours before HTR was transferred via ambulance to ACH. Tr. at 18-19. While at BHMC, HTR's left leg started going numb, and, at the time of transfer, her foot was numb but she had some sensation in the rest of the leg. Tr. at 19. As she was wheeled into her room at ACH a paramedic tested her ability to feel pain with a needle and there was no reaction in either leg. Tr. at 20.

d. Parties' Post Hearing Briefing.

On February 6, 2013, petitioners filed their post hearing brief. The opening paragraph signaled the shift from Dr. Becker's spinal cord infarction theory back to Dr. Kinsbourne's ATM theory: "Dr. Becker originally opined that [HTR] had a spinal cord infarction. At trial however, he acknowledged that if the timing of symptom onset to nadir was longer then he agreed with Dr. Kinsbourne's conclusions rather than Dr. Sladky's conclusions." Pet. Post Hearing Brief at 1. Relying on Mrs. Raymo's

testimony[19] and Pet. Ex. 57, petitioners contend that the onset of HTR's symptoms was at the time she went to bed on the night of October 16, 2013, as it was the last time she was neurologically normal. *Id.* at 9. This would make the period between onset and nadir significantly longer than four hours.[20] Therefore, petitioners maintain that the only logical conclusion is that "HTR's paralysis was the result of an autoimmune condition as opposed to an infarct in her spinal cord." Pet. Post Hearing Brief at 9. Although they did not explicitly withdraw Dr. Becker's expert report, it appears that petitioners are no longer relying on Dr. Becker's HPV-SCI causation opinion.

Respondent's post hearing response, filed February 22, 2013, argued that HTR suffered a spinal cord infarction. Res. Post Hearing Response at 1-2. Stressing Dr. Sladky's strong academic background and clinical experience, particularly as compared to that of Dr. Kinsbourne, respondent urges me to place more weight on his opinion regarding HTR's onset of symptoms and type of injury.

Additionally, with regard to onset of HTR's symptoms, respondent argued that Mrs. Raymo's testimony, which Dr. Kinsbourne relied on in proposing his causation opinion, contradicted the progression of events contained in the contemporaneous medical records. Furthermore, even if onset to nadir took longer than four hours, respondent indicates that her position regarding the type of injury would not change. Res. Post Hearing Brief at 7.

C. Factual Findings Regarding Onset of Symptoms.

With regard to the time elapsed between HTR's vaccination and the earliest symptoms of her transverse myelopathy, and between the earliest symptoms and nadir, I make the following factual findings:

1. HTR received her Tdap vaccination at approximately 11:30 AM on Wednesday, October 13, 2010.

2. It is impossible to conclude precisely when the first symptoms of numbness and tingling in HTR's legs occurred, but I find that they most probably arose while HTR slept, placing onset sometime between 9:30 PM on the evening of Saturday October 16 and before she awoke on Sunday October 17, 2010. Given the pace at which her

[19] Petitioners urge me to place more weight on the testimony of Mrs. Raymo than on the medical records when determining when HTR's symptoms began, noting that the summary of events contained in the records were based on statements made when Mrs. Raymo was "very worried, confused, and stressed." *Id.* at 3.

[20] Doctor Kinsbourne testified that nadir occurred at ACH, when HTR was paralyzed in both legs and had lost all bowel and bladder control. Tr. at 40-41 ("[HTR] cannot move her legs and has trouble with the muscle of the lower body. . . . She can't pass urine in the normal way. She has to be catheterized, and she has great trouble with her bowels . . . [N]adir is when that full set of disabilities first appeared.").

symptoms progressed after awakening, onset most like occurred within an hour or two before awakening.

a. HTR likely slept heavily that evening. She had been up most of Friday night at a church lock-in, followed by a busy day of sightseeing with her parents and grandparents.

b. HTR was still sleeping when her mother awoke in the same room between 7:30-8:00 AM on Sunday, October 17, 2010. Her mother left the room to begin getting ready for church.

c. HTR was awake and sitting on the bed when her mother returned to the room, and she complained to her mother that her right leg was tingling. She also complained of flank pain.

d. Upon Mrs. Raymo's second return to HTR's room that morning, HTR reported that she was not playing, and could not feel her right leg. Mrs. Raymo pinched her daughter's leg and confirmed she could not feel pain.

e. Her parents tried to get her to stand but she was unable to do so. They transported her to the family car in a computer chair and took her to the emergency room at BHMC.

f. HTR continued to lose sensation in her right leg between awaking and her arrival at BHMC around 9:30 AM on October 17, 2010. Numbness in her left leg became more advanced during the car ride, but she still had some sensation of pain in her left leg when examined in the emergency room at BHMC by Dr. Hall.

g. By the time HTR was examined at ACH by Dr. Walters, she was completely paralyzed to the umbilicus. She had lost bowel and bladder control at that point and was experiencing stomach and right flank pain. Given that HTR did not arrive at ACH until 12:25 PM, it is likely that Dr. Walters did not see HTR until close to 1:00 PM, given the need to get her from the ambulance and into an examination room and to take vital signs.

h. Mrs. Raymo's affidavit was filed with the petition. In the affidavit, Mrs. Raymo stated that HTR had weakness and tingling upon awakening. Her affidavit was consistent with her testimony at the hearing. The significance of a short time frame between onset of symptoms and nadir did not arise until Dr. Sladky's report was filed, and the theory that HTR had experienced an SCI was presented.[21] Thus, Mrs. Raymo's affidavit was made before any motive to misrepresent the timeline of events was

[21] Exactly when Dr. Kinsbourne determined that a timeline longer than four hours between onset and nadir could support his original ATM theory is not clear, but it was certainly after Mrs. Raymo's affidavit was prepared and filed.

present. Mrs. Raymo's affidavit is also consistent with the history taken by Dr. Walters, the neurologist who first evaluated HTR at ACH. He recorded that HTR "awoke with numbness of right leg." Pet. Ex. 7, p. 178.

i. Although many of the histories taken at the two hospitals suggest that HTR was "fine" when she awoke, those histories are not inconsistent with the onset of more subtle TM symptoms. The earliest reports note that HTR complained of "flank pain" and numbness. Until HTR tried to stand, the numbness or tingling was likely taken as the common occurrence of "pins and needles" or a limb that is "asleep." It was the inability to feel pain at all and to bear weight that alarmed the Raymos and sent them in panic to the hospital. These more severe symptoms were what Mrs. Raymo and HTR focused on in talking with her doctors.

j. Feeling "fine" on awakening is not inconsistent with TM onset prior to awakening. Attempting to move a tingling extremity and finding it difficult to do so is not inconsistent with not feeling ill or being in pain on awakening.

k. If anyone questioned HTR closely about the precise onset of the numbness or flank pain, it is not apparent from the summarized histories in the medical records. The histories provide more detail about the ascending flaccid paralysis than whether any symptoms first were present upon awakening, upon movement, or upon attempting to bear weight.

3. I find that the time period between onset of symptoms and nadir was longer than four hours, but it is impossible to determine the period precisely. Nadir occurred sometime between 12:30 and 1:00 PM, and onset most likely occurred before 8:00 AM.

4. The time period between vaccination and initial onset of symptoms of a transverse myelopathy is impossible to determine precisely. However, it is more likely than not that the period is between 82 and 93 hours post vaccination, based on the time HTR went to bed on the evening of October 16 and when she likely awoke on the morning of October 17. This places onset at between 3 and 4 days post vaccination.

III. Legal Standards Applying to Off-Table Causation Cases.

When a petitioner alleges an off-Table injury, eligibility for compensation is established when, by a preponderance of the evidence, petitioner demonstrates that he received, in the United States, a vaccine set forth on the Vaccine Injury Table ["Table"] and sustained an illness, disability, injury, or condition caused by the vaccine or experienced a significant aggravation of a preexisting condition. She must also demonstrate that the condition has persisted for more than six months.[22] Vaccine

[22] Section 13(a)(1)(A). This section provides that petitioner must demonstrate "by a preponderance of the evidence the matters required in the petition by section 300aa–11(c)(1) . . . ." Section 11(c)(1) contains the factors listed above, along with others not relevant to this case.

litigation rarely concerns whether the vaccine appears on the Table, the situs for administration, or whether the symptoms have persisted for the requisite time. In most Vaccine Act litigation, the issue to be resolved by the special master is whether the injury alleged was caused by the vaccine. This case is no exception.

To establish legal cause in an off-Table case, Vaccine Act petitioners must establish each of the three *Althen* factors by preponderant evidence: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a proximate temporal relationship between vaccination and injury. *Althen v. Sec'y, HHS,* 418 F.3d 1274, 1278 (Fed. Cir. 2005); *see de Bazan v. Sec'y, HHS*, 539 F.3d 1347, 1351-52 (Fed. Cir. 2008); *Caves v. Sec'y, HHS,* 100 Fed. Cl. 119, 132 (2011), *aff'd per curiam*, 463 Fed. Appx. 932, 2012 WL 858402 (Fed. Cir. 2012) (specifying that each *Althen* factor must be established by preponderant evidence). The applicable level of proof is the "traditional tort standard of 'preponderant evidence.'" *Moberly v. Sec'y, HHS*, 592 F.3d 1315, 1322 (Fed. Cir. 2010) (citing *de Bazan*, 539 F.3d at 1351; *Pafford v. Sec'y, HHS*, 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Capizzano v. Sec'y, HHS*, 440 F.3d 1317, 1320 (Fed. Cir. 2006); *Althen,* 418 F.3d at 1278). The preponderance standard "requires the trier of fact to believe that the existence of a fact is more probable than its nonexistence." *In re Winship*, 397 U.S. 358, 371 (1970) (Harlan, J., concurring) (internal quotation and citation omitted).

Another formulation of the causation requirement in off-Table cases is the "Can it cause?" and "Did it cause?" inquiries used in toxic tort litigation. These queries are also referred to as issues of general and specific causation. Prong 1 of *Althen* has been characterized as an alternative formulation of the "Can it cause?" or general causation query. Prong 2 of *Althen*, the requirement for a logical sequence of cause and effect between the vaccine and the injury, has been characterized as addressing the "Did it cause?" or specific causation query. *See Pafford v. Sec'y, HHS*, No. 01-165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *aff.*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (2006). The third *Althen* factor is subsumed into the other inquiries. Even if a particular vaccine has been causally associated with an injury, petitioner must still establish facts and circumstances that make it more likely than not that this vaccine caused his particular injury. Timing may be one of those circumstances.

Whether a case is analyzed under *Althen* or the "Can it cause?" formulation, petitioners are not required to establish identification and proof of specific biological mechanisms, as "the purpose of the Vaccine Act's preponderance standard is to allow the finding of causation in a field bereft of complete and direct proof of how vaccines affect the human body." *Althen*, 418 F.3d at 1280. The petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the injury or condition; showing that the vaccination was a "substantial factor"[23] in causing the

[23] The Restatement (Third) of Torts has eliminated "substantial factor" in the factual cause analysis. § 26 cmt. j (2010). Because the Federal Circuit has held that the causation analysis in the Restatement

condition and was a "but for" cause are sufficient for recovery. *Shyface*, 165 F.3d at 1352; *see also Pafford,* 451 F.3d at 1355 (petitioner must establish that a vaccination was a substantial factor and that harm would not have occurred in the absence of vaccination). Petitioners cannot be required to show "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1325. Causation is determined on a case by case basis, with "no hard and fast *per se* scientific or medical rules." *Knudsen v. Sec'y, HHS,* 35 F.3d 543, 548 (Fed. Cir. 1994). Close calls regarding causation must be resolved in favor of the petitioner. *Althen*, 418 F.3d at 1280; *but see Knudsen*, 35 F.3d at 550 (when evidence is in equipoise, the party with the burden of proof fails to meet that burden).

By specifying petitioners' burden of proof in off-Table cases as the preponderance of the evidence, directing special masters to consider the evidence as a whole, and stating that special masters are not bound by any "diagnosis, conclusion, judgment, test result, report, or summary" contained in the record (§13(b)(1)), Congress contemplated that special masters would weigh and evaluate opposing expert opinions in determining whether petitioners have met their burden of proof.[24] In weighing and evaluating expert opinions in Vaccine Act cases, the same factors the Supreme Court has considered important in determining their admissibility provide the weights and counterweights. *See Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 149-50 (1999); *Terran v. Sec'y, HHS*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). As the Supreme Court has noted, a trial court is not required to accept the *ipse dixit* of any expert's medical or scientific opinion, because the "court may conclude that there is simply too great an analytical gap between the data and the opinion proffered." *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997).

Although *Daubert v. Merrell Dow Pharmaceuticals*[25] interpreted Federal Rule of Evidence 702, an evidentiary rule not applicable to Vaccine Act cases, *Daubert* nevertheless provides a useful framework for evaluating scientific evidence in Program cases. *Terran*, 195 F.3d at 1316 (concluding it was reasonable for the special master to use *Daubert* to evaluate the reliability of an expert's testimony); *Cedillo v. Sec'y, HHS,* 617 F.3d 1328, 1339 (Fed. Cir. 2010) (noting that special masters are to consider all

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(Second) of Torts applies to off-Table Vaccine Act cases (*see Walther v. Sec'y, HHS*, 485 F.3d 1146, 1151 (Fed. Cir. 2007); *Shyface v. Sec'y, HHS*, 165 F.3d 1344, 1352 (Fed. Cir. 1999)), this change does not affect the determination of legal cause in Vaccine Act cases: whether the vaccination is a "substantial factor" is still a consideration in determining whether it is the legal cause of an injury.

[24] *See* § 13(a)(1)(A) (preponderance standard); § 13(a)(1) ("Compensation shall be awarded . . . if the special master or court finds on the record as a whole . . . ."); § 13(b)(1) (indicating that the court or special master shall consider the entire record in determining if petitioner is entitled to compensation and special master is not bound by any particular piece of evidence).

[25] 509 U.S. 579 (1993).

relevant and reliable evidence filed in a case and may use *Daubert* factors in their evaluation of expert testimony); *Davis v. Sec'y, HHS*, 94 Fed. Cl. 53, 67 (2010) (describing the *Daubert* factors as an "acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted . . . by special masters in vaccine cases"); *see also Snyder v. Sec'y, HHS*, 88 Fed. Cl. 706, 718 (2009) (quoting *Ryman v. Sec'y, HHS*, 65 Fed. Cl. 35, 40-41 (2005) (special masters perform a gatekeeping function when determining "whether a particular petitioner's expert medical testimony supporting biological probability may be admitted or credited or otherwise relied upon" and as a "trier-of-fact [a special master] may properly consider the credibility and applicability of medical theories")). The special master's use of *Daubert*'*s* factors to evaluate the reliability of expert opinions in Vaccine Act cases has been cited with approval by the Federal Circuit more recently in *Andreu v. Sec'y, HHS,* 569 F.3d 1367, 1379 (Fed. Cir. 2009) and *Moberly,* 592 F.3d at 1324. *See also Vaughan v. Sec'y, HHS,* 107 Fed. Cl. 212, 222 (2012) ("The Federal Circuit has repeatedly stated that the Special Master may refer to *Daubert* to assess reliability of expert testimony in vaccine cases."). Special masters decide questions of credibility, plausibility, probability, and reliability, and ultimately determine to which side the balance of the evidence is tipped. *See Pafford*, 451 F.3d at 1359.

IV. Concerns Regarding Expert Credibility.

A. Doctor Becker.

There is preponderant evidence that Dr. Becker plagiarized his expert report from one authored by Dr. Douglas Kerr and filed in another Vaccine Act case. When asked questions about how his report was prepared, Dr. Becker's answers were deliberately misleading. When an expert witness attempts to mislead the court on an issue as fundamental as the origin of his expert opinion, I assess his credibility as so severely compromised as to preclude reliance upon his opinion and testimony.

The issue of whether Dr. Becker's report was his own first arose during the entitlement hearing. During cross-examination of Dr. Becker, respondent's counsel inquired as to the process Dr. Becker used to draft the expert report in this case. Tr. at 140-141. He indicated that usually he "get[s] the records provided by the patients, by the offices, and then [he] review[s] those and start[s] forming [his] opinion after complete review of those records." Tr. at 140. With regard to this case, he testified that he:

> had pretty specific things to cover. The timeline supports the evidence, and so I had to find papers in the literature that backed up the statements that I had made that I got to after reviewing the report. So generally, like scientific papers, and Dr. Kinsbourne probably knows that, and Dr. Gill, anybody of us who writes scientific papers, you start with your findings and in your discussion you try to be all-inclusive, trying to find the appropriate background information to make sure you're appropriately covered and you can support your claims.

Tr. at 141. Respondent's counsel then asked Dr. Becker if he had consulted with Dr. Douglas Kerr, a founder of the clinic at which Dr. Becker works, while preparing his report. *Id.* Doctor Becker indicated that although he knew Dr. Kerr personally, he had not consulted with him when drafting his expert report. *Id.*

At the beginning of her direct examination, Dr. Gill testified that she had consulted on about six cases in the Vaccine Program. In connection with one of those cases, she had reviewed a report written by Dr. Kerr which was "extremely similar" to the report filed by Dr. Becker in this case. Tr. at 311-12. She testified: "[A] large proportion of [Dr. Becker's] report is word-for-word what Dr. Kerr had written for the other case and all of the references were the same." Tr. at 312. Doctor Gill also testified that, other than the name of petitioner, the two reports contained identical paragraphs, including identical emphasizing of particular words in the text.[26]

Post hearing, respondent filed a status report addressing the issue of plagiarism in Dr. Becker's expert report. Respondent asserted that Dr. Gill had alerted respondent's counsel to the similarities between the two reports prior to the hearing, which is why counsel inquired as to Dr. Becker's process for preparing expert reports. Respondent's Status Report, filed Jan. 31 ["January Status Report"], 2013, at 1-2. Because § 12(d)(4)(A) prevented respondent from disclosing the contents of Dr. Kerr's report to a party not involved in the case in which it was filed, respondent's counsel had sought permission from petitioner's counsel in the similar case (*Flores v. Sec'y, HHS*, No. 10-489V) to file a redacted version of Dr. Kerr's expert report into the record of this case. January Status Report at 3. However, counsel for petitioner in *Flores* indicated that his client would not consent to the disclosure of a redacted version of Dr. Kerr's report. *Id; see also* Respondent's Status Report, filed Feb. 8, 2013.

Respondent's counsel asserted she had read Dr. Kerr's report in both cases and confirmed that Dr. Gill's testimony regarding the two reports was accurate. January Status Report at 2 (noting that entire paragraphs were identical and seventeen of exhibits cited in Dr. Becker's report were cited by Dr. Kerr in *Flores*). Additionally, respondent noted that Dr. Kerr's report was dated February 21, 2011, and therefore was prepared eight months prior to the filing of this petition and more than a year before petitioners filed Dr. Becker's expert report.

Petitioners did not directly address respondent's allegations of plagiarism by Dr. Becker. However, in their post hearing brief, filed after respondent brought her

[26] Tr. at 312. On cross-examination, Dr. Gill could not recall the name of the other case, but she did indicate that it was heard by Special Master Hastings, involved a child treated at Rush in Chicago, and, similar to this case, alleged a vascular event related to a Gardasil vaccination. Tr. at 339-40. The *Flores* case involves a child treated at Rush for a purported vascular event after a Gardasil vaccination, and was heard by Special Master Hastings. *Flores v. Sec'y, HHS*, No. 10-489V, 2013 WL 5587390, at *1, 3, 6 (Fed. Cl. Spec. Mstr. Sept. 12, 2013).

concerns to the court's attention, petitioners abandoned the HPV-SCI causation theory Dr. Becker proposed and returned to their initial Tdap-ATM causation theory.

I have compared Dr. Becker's report with the portions of Dr. Kerr's report quoted in *Flores*, and the portions quoted are virtually identical. *See Flores*, 2013 WL 5587390, at *6 n.11, *12 n.16, *14 n.19.

It is clear that Dr. Becker presented the work product of Dr. Kerr as his own. It does not appear that he disclosed this fact to petitioners or their attorney, at least before the issue was raised at hearing. It is this failure that I find the most concerning in deciding whether to credit any part of his testimony. Had Dr. Becker indicated that he had been provided a copy of Dr. Kerr's earlier report and agreed with the reasoning and conclusions therein and adopted them as his own, my concerns about his candor would be less pressing. However, whether for financial reasons, time constraints, or for the prestige attached to being an expert witness,[27] Dr. Becker was willing to take a shortcut, pass another's work product off as his own and, more significantly, testify in a manner that attempted to mislead the court about the origin of the opinions expressed in the report bearing his signature.

B. Doctor Sladky.

Unfortunately, witnesses with ethical challenges were a problem for both parties. Respondent took a more forthright approach to Dr. Sladky's licensure problems by bringing them to the court's attention. On May 1, 2013, respondent filed a status report concerning Dr. Sladky. In summary, the status report informed the court that respondent had recently learned that Dr. Sladky had withheld information regarding suspension of his license to practice medicine. May Status Report at 1. Although he was properly licensed throughout his involvement with this case, he neglected to disclose to respondent a prior suspension of his medical license and the completion of a probationary period. *Id.* Respondent attached several documents to her status report from the Georgia Composite Medical Board ["GCMB"] detailing the specifics of Dr. Sladky's suspension and probation.[28]

Petitioners filed a response to respondent's status report on May 6, 2013. The response noted that petitioners "do not wish to delay a ruling by the Special Master in this matter [and] thus will not file a formal objection at this time," but wanted to "make a

[27] He testified that this case was his first appearance as an expert witness. Tr. at 96.

[28] It appears from respondent's status report that Dr. Sladky prepared expert opinions during the period in which his license to practice medicine was suspended and/or in which he was practicing under supervision. May Status Report at 2; s*ee also Contreras v. Sec'y, HHS*, No. 05-626V, 2013 WL 6698382, at *4-5 (Fed. Cl. Spec. Mstr. Nov. 19, 2013) (discussing Dr. Sladky's supplemental expert report and hearing testimony in relation to the end of his suspended license and start of probationary period in March 2010).

note on the Record that Petitioners were not given the opportunity to question Dr. Sladky regarding his suspension/probation." Reply to Respondent's Status Report at 1. During a status conference, held on May 17, 2013, I stressed that any issue petitioners might want to raise in a future motion for review must be raised before the special master. Order, issued May 17, 2013, at 2. I offered petitioners the option of recalling Dr. Sladky for additional cross-examination or propounding interrogatories for him to answer. Additionally, I indicated that petitioners could propose other methods for addressing their concerns. *Id.* I also noted, that based on my workload, supplemental briefing or proceedings would not likely delay an entitlement decision. Petitioners declined the opportunity I afforded them to pursue this issue further. Petitioners' Response to May 17, 2013 Order, filed May 22, 2013, at 2 ("[C]ounsel for Petitioners take the position that the matter should be left to the Special Master's evaluation and resolution without the need for further action by Petitioners.").

In light of respondent's disclosures, I looked carefully at the testimony of Dr. Sladky and his CV, filed as Res. Ex. B. Although filed with his expert report in February 2012, his CV is dated January 5, 2009. His CV therefore was written or updated near the end of the period during which Dr. Sladky had agreed not to practice medicine. Attachment to May Status Report at 2-3 (March 2010 Public Consent Order between Dr. Sladky and the GCMB). However, Dr. Sladky's CV does not reflect that he had taken leave from his hospital appointments.

Doctor Sladky was even more careful than Dr. Becker to avoid perjuring himself. He testified that he began working at Emory University in 1995 and had recently retired and moved to a private practice in Atlanta. Tr. at 187. He did not mention that between 1995 and 2012 there were periods when he had a suspended medical license or practiced only on a probationary basis. When asked to describe his day-to-day activities while at Emory and in his current position, he carefully prefaced his answer with "when I was on service." Tr. at 188. This preface could reflect the difference in his roles when performing medical duties versus his administrative or teaching duties. Alternatively, it could be considered a carefully crafted answer to avoid giving perjured testimony. By specifying that his answer pertained to the time periods when he was practicing medicine, he avoided the necessity of indicating that there were periods when he was not able to practice medicine due to the suspension of his medical license.

Standing alone, the basis for Dr. Sladky's disciplinary action might not affect the reliability of his expert opinions. However, his failure to disclose the disciplinary action to respondent, his authoring of expert opinions while he did not have an active medical license, and the failure to reflect his voluntary leave from medical practice due to a substance abuse problem on the CV filed in this case all cast doubt about his credibility as a witness.

C. Discussion.

The situation of an expert plagiarizing from another report prepared by a colleague appears to be one of first impression in the Vaccine Program. When faced with a similar situation, now Chief Judge Sarah Vance of the Eastern District of Louisiana wrote:

> Using the opinions of another does not automatically render expert testimony inadmissible. *See e.g., Legier and Matterne v. Great Plains Software, Inc.,* 2005 WL 2037346, at *4 (E.D.La. Aug. 3, 2005) (denying motion to strike testimony based in part on allegations that paragraph in expert report was plagiarized). Yet, here, Dr. Kura's use of Dr. Kopstein's work is particularly problematic in that Dr. Kura first testified that the report he proferred was his original drafting and that he had not reviewed other expert reports. When asked to explain why many of his sentences were nearly identical to Dr. Kopstein's, he later conceded that he saw Dr. Kopstein's report and at the very least took notes. The likelihood that substantial portions of Dr. Kura's report do not reflect his original work is yet another reason[29] the Court finds that Dr. Kura's opinions in general are unreliable. The Court therefore deems Dr. Kura's report and testimony to be inadmissible at trial in their entirety.

Moore v. BASF Corp. 2012 WL 6002831, at *7 (E.D.La. 2012) (footnotes omitted).

"Resume malfunctions" were discussed with regard to at least one witness during the Omnibus Autism Proceeding test case hearings. *See Snyder v. Sec'y, HHS*, No. 01-162V, 2009 WL 332044, *14-15 (Fed. Cl. Spec. Mstr. Feb. 12, 2009) (discussing Dr. Byers' resume and its exaggerated descriptions of her past work experience). I was unable to find any prior Program decisions that specifically addressed the impact of filing a CV or providing testimony that represented an expert was licensed and practicing medicine during periods when he was not.

When considering a motion to exclude expert testimony, Magistrate Judge Kravchuk concluded that an individual whose CPA license had expired was qualified to opine as an accounting expert. However, she noted that the "factfinders will be allowed to hear about [the expert's] difficulties with the licensing authority and that in spite of his license being suspended, he described himself as a CPA. A factfinder might well decide to give his opinion little weight in light of his professional difficulties, or not." *Fitzpatrick v. Teleflex, Inc.* 763 F.Sup.2d 224, 236 (D. Me. 2011).

[29] The other reasons for rejecting Dr. Kura's report concerned the methodology he used to estimate the amount of benzene in the defendant's product and the conditions and number of hours worked by plaintiff, which impacted the exposure calculation.

In *Moberly*, the Federal Circuit stated that "[f]inders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence." 592 F.3d at 1326. When evaluating inaccurate testimony, there is a significant difference between forgetfulness and a person making a deliberate decision to mislead. *See Cardiac Pacemakers, Inc., v. St. Jude Medical, Inc.*, No. IP 96-1718-Ch/K, 2002 WL 1801525, at *61 (S.D. Ind. 2002) (describing the difference as a "wide chasm" and noting a juror's "trust or confidence in a witness's honesty can be critical").

Although both Dr. Becker and Dr. Sladky are well qualified to opine, I cannot rely on their opinions.[30] I administer an oath to witnesses that requires that they tell the whole truth. Neither Dr. Becker nor Dr. Sladky told the whole truth. Both demonstrated a lack of candor that, although not related directly to the substance of their causation opinions, reflect their willingness to, at the very least, shade the truth. In the case of Dr. Becker, he attempted to pass off another's work as his own. In the case of Dr. Sladky, it appears that he so feared the loss of his position and income as a case reviewer for respondent that he withheld facts concerning his medical license suspension. I thus do not rely at all on their expert opinions in this case.

V. Analysis of Causation Evidence.

A. Overview.

Petitioners are relying exclusively on the ATM diagnosis and an autoimmune theory of causation as set forth in Dr. Kinsbourne's supplemental expert report (Pet. Ex. 9) and testimony. Additional support for HTR's diagnosis is found in Dr. Barnes' expert report (Pet. Ex. 29)[31] and in the opinions of HTR's treating physicians. Support for Dr. Kinsbourne's causation theory is found in the medical literature filed and in a "second opinion" obtained for purposes of diagnosis and treatment, rather than this litigation.

Because I attach no weight to the opinions of Drs. Sladky and Becker, Dr. Kinsbourne's opinion is largely unrebutted. Although I have considered the expert report and testimony of Dr. Gill, her evidence was almost exclusively focused on

[30] I am aware that two of my colleagues have also considered the import of Dr. Sladky's problems on the reliability of his opinions. *Contreras*, 2013 WL 6698382, at *4-5 (accepting Dr. Sladky's opinion on diagnosis, as it was supported by the opinion of treating physicians) ; *Roberts v. Sec'y, HHS*, No. 09-427V, 2013 WL 5314698, at *9 (Aug. 29, 2013) (rejecting Dr. Sladky's opinion as unreliable).

[31] Doctor Barnes' opinion is not particularly relevant to a specific cause once spinal cord infarction is no longer part of the causation equation because it addressed only the diagnostic significance of the inflammation.

demonstrating that Dr. Becker's theory regarding a vascular cause for HTR's infarction was unsound,[32] and thus is not relevant to the causation theory still before me.

Cross-examination identified some issues with both Dr. Kinsbourne's qualifications and his causation opinion. Nevertheless, petitioners have established by preponderant evidence that the correct diagnosis is acute immune-mediated transverse myelitis, that the medical theories proposed are biologically probable, that the facts and circumstances surrounding HTR's vaccination and onset establish a logical connection between vaccine and injury, and that the timing between these two events is medically appropriate.

The threshold issue in this case—the length of time between onset and nadir—was resolved by my factual findings. This resolution brings Dr. Kinsbourne's expert opinions into play, because he candidly acknowledged that if the period between onset and nadir was shorter than four hours, HTR could not have ATM, based on the diagnostic criteria for that condition. Tr. at 37-38. With an onset longer than four hours, I find that HTR meets the diagnostic criteria for ATM.[33]

The pivotal issue then becomes whether there is preponderant evidence that HTR's ATM was caused by the tetanus component of her Tdap vaccination. The very unusual circumstances pertaining here lead me to conclude that petitioners have met their burden to so demonstrate.

B. Credibility and Reliability Issues.

Doctor Kinsbourne is not an ideal expert witness. Not only does he derive a substantial proportion of his income from his employment as an expert witness,

[32] Doctor Gill's primary role in the case was to "review [HTR's] records and respond to Dr. Becker's opinion regarding the possible cause of her injuries in relation to her HPV vaccine." She was not asked to "respond to any other part of the case or any other vaccine." Tr. at 310; *see also* Res. Ex. J at 1.

[33] The diagnostic criteria are: (1) bilateral sensorimotor and autonomic spinal cord dysfunction; (2) clearly defined sensory level; (3) progression to nadir of clinical deficits between 4 hours and 21 days after symptom onset; (4) demonstration of spinal cord inflammation; (5) exclusion of compressive, post-radiation, neoplastic, and vascular causes. Frohman and Wingerchuk, Pet. Ex. 11, at 565 (Table 1). The Transverse Myelitis Consortium Working Group also included the exclusion of a compressive etiology, spinal radiation, clear arterial distribution deficit, connective tissue disease, multiple sclerosis, evidence of infection in the central nervous system and optic neuritis. TMCWG Diagnostic Criteria, Res. Ex. D, at 500 (Table 1). The only diagnostic criterion in question was the time period between nadir and onset. Spinal cord inflammation was demonstrated via spinal cord MRI, including a gadolinium-enhancing cord lesion found during the acute phase of HTR's symptoms. Pet. Ex. 25, p. 1345. Although the lumbar puncture did not demonstrate inflammation, the evidence was that it was performed too soon after onset to contain evidence of inflammation, and a subsequent lumbar puncture was never performed. *See* Pet. Ex. 7, pp. 189, 203; Tr. at 54. I note that lesions associated with idiopathic transverse myelitis usually span at least two vertebral segments, and HTR's spanned about four segments, with abnormal signal from the T8-9 to the T11-12 levels. Pet. Ex. 25, p. 1345.

primarily in the Vaccine Program (Tr. at 46) but he does not treat transverse myelopathies, let alone teach, research, or write about them (*see* Tr. at 46-48). He has not engaged in the clinical practice of medicine for more than three decades. Tr. at 46. However, he is a licensed physician and one trained in a discipline, pediatric neurology, relevant to the condition from which HTR suffers, and is thus qualified to offer an opinion on causation. In the absence of any contrary opinion on which I can rely, it is also a persuasive opinion.

The assertions made in Dr. Kinsbourne's written opinions[34] are not merely his *ipse dixit*. They are supported by the medical literature filed. His conclusion on causation is supported by the similar opinion of Dr. Mateen, a post-doctoral fellow at Johns Hopkins, who was asked to render a medical opinion on HTR's condition, not an expert opinion for purposes of litigation.[35] Doctor Mateen concluded that "the most likely etiology for [HTR]'s symptoms is transverse myelitis . . . as a rare complication of vaccination." Pet. Ex. 54, p. 1415.

C. The Specifics of Dr. Kinsbourne's Causation Opinion.

Doctor Kinsbourne's expanded causation opinion, filed as Pet. Ex. 9, can be summarized as follows.

1. Diagnosis.

HTR met the diagnostic criteria for ATM. Pet. Ex. 9, p. 2. The MRI performed during the first day of symptoms showed an abnormal signal, suggestive of inflammation. *Id*. at 2; Tr. at 54, 72-73.

2. Cause of ATM in General.

ATM is "an acute focal inflammatory disorder of the spinal cord." Pet. Ex. 9 at 2. Direct nervous system infection is rare in TM or other nervous system disorders. *Id*. at 3. In TM, the lesions in the spinal cord are caused by inflammation; pathogens themselves are nearly always absent. *Id.* at 5. The lesions are the result of an immune-mediated attack on self-antigens, but the precise biological mechanism by which this attack occurs is unknown. There is evidence that a proinflammatory cytokine, IL-6, in the spine plays a causal role in the lesions. What triggers the increased levels of IL-6 in the spinal cord is unknown, but there is evidence of T-cell

[34] His testimony was much less specific than his written opinions, found at Pet. Exs. 1 and 9.

[35] In 2012, Mr. and Mrs. Raymo contacted the Johns Hopkins Transverse Myelitis Center to seek potential treatment for HTR. To begin the evaluation process, they were asked to send medical records for the facility's Remote Second Opinion Program. Doctor Mateen was the physician assigned to review HTR's records and author a report regarding her injury. *See* Petitioners' Notice of Intent to File Medical Records, filed July 10, 2012.

activation, and an immune response to vaccination or infection, which are considered candidates for this activation. *Id*. at 4-5; Tr. at 70-71. ATM can be triggered by many pathogens; the precise cause cannot be determined by the clinical symptoms presented. Pet. Ex. 9 at 3.

3. Tetanus Vaccine Causation of ATM.

The proposition that vaccine antigens in general and the tetanus toxoid in particular can cause ATM is relatively well accepted in the medical literature. Pet. Ex. 9 at 3-4; Tr. at 42-43, 59. Tetanus toxoid is recognized as capable of inducing immune-mediated neurological disorders and ATM is usually an immune-mediated disorder. Pet. Ex. 9 at 4-5. Numerous case reports involving tetanus vaccinations and subsequent ATM support a causal relationship. *Id.* at. 4; Tr. at 66-68.

4. Timing.

The temporal interval between vaccination and symptom onset in this case is medically reasonable for an immune mediated disorder, particularly in view of the five earlier tetanus toxoid-containing vaccinations that HTR had received. The speed of reaction is based on an anamnestic response. Pet. Ex. 9 at 6; Tr. at 32-34, 55-57.

5. Lack of Alternate Cause.

Although not necessary, Dr. Kinsbourne also addressed the issue of a possible alternate cause. HTR was assessed as having a URI at the time of her vaccination, but her symptoms were consistent with allergic rhinitis rather than an infection. He concluded that HTR had allergies, not an acute illness, at the time of her vaccination, based on the lack of fever, purulent discharge, or sore throat, and the similarity of her symptoms to previous bouts of allergic rhinitis. Pet. Ex. 9 at 1, 6. Thus, Dr. Kinsbourne discounted any antecedent infection as an alternate clause. *Id*. at 6.

6. Conclusion.

Doctor Kinsbourne concluded that the tetanus toxoid component of the [Tdap][36] vaccination could and did cause HTR's ATM, to a reasonable degree of medical probability. Pet. Ex. 9 at 6.

[36] His report incorrectly refers to the causal vaccination as a DTaP vaccination. Pet. Ex. 9 at 6. Although this is without significance in this case, the lack of precision in his report does not enhance the reliability of his opinion.

D. Support for Dr. Kinsbourne's Opinions and Conclusions.

1. Causes of ATM in General.

Identifying the cause of transverse myelitis is often challenging and, in many circumstances, the cause remains unknown. Frohman and Wingerchuck, Pet. Ex. 11, at 571. About 15-30% of cases are classified as idiopathic, meaning no cause is determined. *Id.* at 564-65. The pathological hallmark of ATM is "the presence of focal collection of lymphocytes and monocytes, with varying degrees of demyelination, axonal injury, and astroglial and microglial activation, within the spinal cord." *Id.* at 564. Forty percent of TM cases are associated with an antecedent infection occurring from days to weeks before onset of symptoms. Because the antecedent viral or bacterial infectious agent is not found in the CSF, the linkage is not likely a directly infectious one. An autoimmune process is likely. N Agmon-Levin, et al., *Transverse myelitis and vaccines: a multi-analysis*, LUPUS, 18: 1198-1204 (2009), filed as Pet. Ex. 10 [hereinafter "Agmon-Levin, Pet. Ex. 10"] at 1199.

Because many cases of ATM are considered to be immune-mediated, molecular mimicry is often considered to be the likely mechanism of injury. The 2012 IOM Report noted that "[a]utoantibodies, T cells, and molecular mimicry may contribute to the symptoms of transverse myelitis." 2012 IOM Report at 471.

2. Molecular Mimicry as a Mechanism of Injury.

Molecular mimicry is defined by the 2012 IOM Report as "sequence and/or conformational homology between an exogenous agent (foreign antigen) and self-antigen leading to the development of tissue damage and clinical disease from antibodies and T cells directed initially against the exogenous agent that also react against self-antigen." 2012 IOM Report at 70.

Although not definitively established as the causal mechanism in any human disease,[37] molecular mimicry is nevertheless used to explain how a variety of infections or vaccinations can cause such nervous system disorders as acute disseminated encephalomyelitis, Guillain-Barré syndrome, and transverse myelitis. In these conditions, there is no evidence of the causal pathogen at the location of the inflammation or demyelination; thus, rather than a direct attack by a pathogen, the immune system is triggered or stimulated into attacking the body's own tissue (often

[37] In Pet. Ex. 13, Drs. Kerr and Ayetey asserted that molecular mimicry was best described in GBS. D Kerr and H Ayetey, *Immunopathogenesis of acute transverse myelitis,* CURR. OPIN. NEUROL., 15 (3): 339-47 (2002) [hereinafter "Kerr and Ayetey, Pet. Ex 13"] at 342. The 2012 IOM Report used rheumatic fever associated with group A streptococcal infection as an example "implicat[ing] this mechanism in certain human autoimmune diseases." 2012 IOM Report at 71. The 2012 IOM Report also described the relationship between antigens from *C. jejuni* and autoantibodies bound to neuronal glangliosides in a specific form of GBS, the same association described in Pet. Ex. 13. *Id.* at 72.

described as "cross-reactivity" or a break in the "self-tolerance" usually exhibited by immune system cells). *See, e.g.*, Kerr and Ayetey, Pet. Ex. 13, at 342 (describing cross-reactivity of antibodies to *C. jejuni* surface antigens and peripheral nerves).

Precisely how this autoimmune attack is precipitated is unclear in general, although some degree of homology between molecular sequence of the pathogen and the myelin sheath of nerves or axons is presumed. In some cases, such homology has been demonstrated (*C. jejuni* in Guillain Barré syndrome, for example). However, in ATM, homology has not been demonstrated between any suspected precipitating agent and the spinal cord nerve sheaths or axons.

One theory that explains this lack of homology is that the autoantibodies themselves cause direct injury to neurons. Another is the bystander activation theory, in which antigens generate a T-cell response, followed by the production of pro-inflammatory cytokines, such as the IL-6 implicated in the Kaplin study, Pet. Ex. 12, discussed below.

3. Bystander Activation.

IL-6 is an inflammatory marker present in CSF in TM and the level of IL-6 correlates with the severity of symptoms. Agmon-Levin, Pet. Ex. 10, at 1198. In one small study, the mean level of IL-6 in CSF was more than 200 times higher in TM patients than in controls. Serum IL-6 levels were not markedly different, indicating that the IL-6 was being generated in the CSF. A. Kaplin, et al., *IL-6 induces regionally selective spinal cord injury in patients with the neuroinflammatory disorder transverse myelitis,* J. CLIN. INVEST., 115:2731-41 (2005), filed as Pet. Ex. 12 [hereinafter "Kaplin, Pet. Ex. 12"] at 2733. In two patients who died from TM, autopsy demonstrated that astrocytes in and around the area of damage were the predominant source of the IL-6 found. *Id*.

A causal role for IL-6 can be inferred from the fact that CSF containing high levels of IL-6 taken from a TM patient induced cell death in spinal cord cells in culture whereas CSF from a control patient did not. Kaplin, Pet. Ex. 12, at 2734. When the IL-6 present in the CSF from the TM patient was removed, the CSF did not induce cell death, suggesting that the IL-6 was causative, rather than a marker for inflammation. *Id*. The presence of IL-6 in post-infectious TM suggests that the biological mechanism involves bystander activation. Agmon-Levin, Pet. Ex. 10, at 1201.

4. Causal Role of Vaccinations.

There are no epidemiologic studies of the causes of ATM, and thus no studies linking or refuting a link between the condition and vaccinations. However, Dr. Kinsbourne's assertions that medical literature acknowledges a causal connection between the two events is correct. For example, a clinical practice article published in 2010 in the New England Journal of Medicine states:

> The transverse myelitis syndrome may arise from various causes, but it most often occurs as an autoimmune phenomenon after an infection or vaccination (accounting for 60% of the cases in children) or as a result of a direct infection, an underlying systemic autoimmune disease, or an acquired demyelinating disease such as multiple sclerosis or the spectrum of disorders related to neuromyelitis optica (Devic's disease, a demyelinating disease that is defined by transverse myelitis and optic neuritis). However, after detailed evaluation, 15 to 30% of the cases are ultimately categorized as idiopathic. . . . The observation that systemic infection or immunization precedes many cases of transverse myelitis suggests that mechanisms such as molecular mimicry and the development of autoantibodies may play roles in the pathogenesis of the syndrome.

Frohman and Wingerchuk, Pet. Ex. 11, at 564-65. A medical textbook, CHILD NEUROLOGY, also reports a connection between vaccinations (including tetanus vaccinations) and TM. J Menkes, et al., CHILD NEUROLOGY, 7th ed., Lippincott, Williams and Wilkins (2006), filed as Pet. Ex. 14, at 587. Doctor Douglas Kerr,[38] the corresponding author for the Transverse Myelitis Consortium Working Group (*see* TMCWG Diagnostic Criteria, Res. Ex. D, at 499 n.*), wrote in 2002 that "it is widely reported in neurology texts that ATM is a post-vaccination event." Kerr and Ayetey, Pet. Ex. 13, at 340. Background information in Res. Ex. D also acknowledges a link between some vaccinations and TM, reporting more than 200 cases of TM in England in 1922-23 as a complication of smallpox and rabies vaccinations. TMCWG Diagnostic Criteria, Res. Ex. D, at 499.

I thus conclude that there is adequate evidence that vaccinations in general can cause ATM. Although the precise biological mechanism has not been determined, molecular mimicry and bystander activation theories are biologically probable.

5. Causal Role for Tetanus Vaccination.

Doctor Kinsbourne identified the tetanus component of HTR's Tdap vaccination as causal because it was the "best documented" in medical literature as stimulating adverse neurological effects. Tr. at 42-43. The 1994 IOM Report indicated that a causal role for tetanus toxoid in central nervous system disease was "biologically plausible." 1994 IOM Report, filed as Pet. Ex. 20, at 85. In discussing transverse myelitis in particular, the 1994 IOM committee found the theory of an autoimmune response induced by vaccination biologically plausible, but the committee did not find any epidemiological evidence sufficient to conclude that vaccinations caused TM. *Id.* at 84.

[38] This appears to be the same Dr. Kerr whose expert report Dr. Becker copied.

Several case reports discussed the temporal link between tetanus-containing vaccinations and TM. S Read, et al., *Acute transverse myelitis after tetanus toxoid vaccination*, LANCET, 339:1111-12 (1992), filed as Pet. Ex. 17; R Riel-Romero, *Acute transverse myelitis in a 7-month-old boy after diphtheria-tetanus-pertussis immunization*, SPINAL CORD, 44:688-691 (2006), filed as Pet. Ex. 18; F Tezzon, et al., *Acute radiculomyelitis after antitetanus vaccination,* ITAL. J. NEUROL. SCI., 15:191-93 (1994), filed as Pet. Ex. 22; H Topaloglu, *Optic neuritis and myelitis after booster tetanus toxoid vaccination*, LANCET, 339:178-79 (1992), filed as Pet. Ex. 23; E Whittle and N Robertson, *Transverse myelitis after diphtheria, tetanus and polio immunization*, BRIT. MED. J., 1(6074): 1450 (1977), filed as Pet. Ex. 24. Additionally, a case series was filed that discussed tetanus and polyneuropathies. S Rutledge and O Snead, *Neurologic complications of immunization*, J. PEDIATR., 109(6): 917-924 (1986), filed as Pet. Ex. 19.

Case reports are not, in general, strong evidence of causation. However, ATM is a relatively rare condition, with only about 1400 new cases in the U.S. diagnosed annually. F Pidcock, *et al.*, *Acute transverse myelitis in childhood: Center-based analysis of 47 cases*, NEUROL., 68:1474-60 (2007), filed as Pet. Ex. 16 [hereinafter "Pidcock, Pet. Ex. 16"] at 1474. About 80% of cases involve adults for whom a tetanus vaccination is recommended only every 10 years. *Id.;* Td Vaccine Information Sheet, available at http://www.cdc.gov/vaccines/hcp/vis/vis-statements/td.html (last visited Feb. 19, 2014). Thus, these case reports of a rare condition, ATM, following a tetanus vaccination in an adult carry more significance than I might otherwise accord them. However, I note that the 2012 IOM Report considered case studies insufficient to show causation. 2012 IOM Report at 548. This approach by the IOM, while informative regarding causation, does not bind special masters, as it is apparent that the IOM requires a very high standard before concluding that there is a causal relationship between vaccines and an injury.[39] The 2012 IOM Report noted that "[a]utoantibodies, T cells, and molecular mimicry may contribute to the symptoms of transverse myelitis," but found inadequate evidence to link tetanus-toxoid-containing vaccines to these mechanisms. 2012 IOM Report at 548.

[39] Doctor Kinsbourne made this argument and I agree with his assertion that the IOM requires a higher standard for concluding causality has been established than the preponderant evidence standard found in the Vaccine Act. I note that, notwithstanding significant evidence that some strains of the influenza virus can and do cause Guillain-Barré syndrome (*see Tompkins v. Sec'y, HHS*, No. 10-261V, 2013 WL 3498652, at *22-23 (Fed. Cl. Spec. Mstr. June 21, 2013)), the 2012 IOM Report concluded that "[t]he evidence is inadequate to accept or reject a causal relationship between influenza vaccine and GBS." 2012 IOM Report at 334. The 2012 IOM Report asserted that "molecular mimicry was not confirmed to be a mechanism leading to the development of the adverse events post-vaccination." *Id*. at 73. The use of the word "confirmed" in this assertion validates Dr. Kinsbourne's claim that the standard of proof required by the IOM was higher than petitioners' burden of proof (preponderant evidence) in Vaccine Act cases. Pet. Ex. 9 at 5-7; Tr. at 60-61.

There is some evidence beyond case studies. Petitioners' Exhibit 16 is a study of ATM in 47 children. In 28% of the cases, vaccination or an allergy shot preceded the initial symptom of ATM within 30 days. At least two of the 13 children received a tetanus-toxoid-containing vaccination. Febrile illness preceded onset in 47% of cases. Pidcock, Pet. Ex. 16, at 1476. The study was focused on the persisting nature of the disability in many children. The authors declined to draw any conclusion regarding vaccine causation. *Id.* at 1474 (Abstract), 1479.

In a multi-analysis of vaccines and transverse myelitis, 37 cases of TM were examined. Most occurred within a month of a vaccination, and the cases were about evenly split between adults and children. Four cases occurred after a tetanus-toxoid-containing vaccination, and one additional case involved a DT vaccination administered concurrently with other childhood vaccinations. Agmon-Levin, Pet. Ex. 10, at 1200 (Table 1). One case involved a 13 year old, with onset of symptoms within three days of vaccination.

In discussing how HTR's treating physicians arrived at their diagnosis, Dr. Kinsbourne explained that myelopathies such as TM have many possible causes. Tr. at 35. These include infection, trauma, tumors, some diseases, and some neurological conditions. Because there are so many possible causes, physicians perform tests to rule out specific diagnoses. HTR's physicians ruled out all of these as causes for her condition. *Id.* That left what Dr. Kinsbourne considered the most common cause of this type of myelopathy, an autoimmune attack on the spinal cord triggered by some agent. *Id*. He agreed with her treating physicians that HTR suffered from acute transverse myelitis, an inflammatory condition of the spinal cord. Tr. at 32.

In cases in which a petitioner has established that the vaccine can cause the injury and the injury arose in the correct time, the exclusion of other factors may be probative that the vaccine caused the injury. A prerequisite is that the petitioner establish, on a more likely than not basis, that the vaccine can cause the injury. *See Contreras*, 2013 WL 6698382, at *58 (citing *Tamraz v. Lincoln Elec. Co.,* 620 F.3d 665, 674 (6th Cir. 2010); *Hendrix ex rel. G.P. v. Evenflo Co., Inc*., 609 F.3d 1183, 1197-98 (11th Cir. 2010); *Ruggiero v. Warner-Lambert Co*., 424 F.3d 249, 254 (2d Cir. 2005)). Doctor Kinsbourne applied this reasoning to determine that HTR's tetanus vaccination did cause her ATM.[40] He concluded that the most likely causal agent in this case was the tetanus component of the Tdap vaccination HTR received on October 13, 2010, because it was the best documented in the medical literature as stimulating adverse neurological effects. Tr. at 42-43.

[40] In contrast, the exclusion of other potential factors does not promote the finding that the vaccine can cause the injury, which is the inquiry in *Althen* prong one. *See Caves*, 100 Fed. Cl. at 144; *Veryzer v. Sec'y, HHS*, 100 Fed. Cl. 344, 355-56 (2011).

Although other decisions involving this vaccine and this injury do not constitute binding authority,[41] Dr. Kinsbourne's opinion is also buttressed by other decisions in the Vaccine Program awarding entitlement for ATM associated with a tetanus vaccine. *See e.g., Roberts v. Sec'y, HHS,* No. 09-427V, 2013 WL 5314698 (Fed. Cl. Spec. Mstr. Aug. 29, 2013); *Helman v. Sec'y, HHS,* No. 10-813V, 2012 WL 1607142 (Fed. Cl. Spec. Mstr. Apr. 5, 2012); *Hargrove v. Sec'y*, *HHS,* No. 05-694V, 2009 WL 1220986 (Fed. Cl. Spec. Mstr. Apr. 14, 2009); *Bowes v. Sec'y*, *HHS,* No. 01-481V, 2006 WL 2849816 (Fed. Cl. Spec. Mstr. Sept. 8, 2006).

In view of the Federal Circuit's opinion in *Capizzano* that petitioners cannot be required to show "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect"*(*440 F.3d at 1325), I rely on the opinions of Drs. Kinsbourne and Mateen that there is a sufficient connection between the theory of causation and the facts in this case to establish the second *Althen* factor.

6. A Medically Appropriate Temporal Relationship.

Petitioners have the burden to demonstrate the existence of a "scientific temporal relationship." *Pafford v. Sec'y, HHS*, 64 Fed. Cl. 19, 29-30 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006). The time frame must be medically acceptable. *de Bazan*, 539 F.3d at 1352. The evidence that the three to four day period between vaccination and onset of symptoms in this case comes primarily from the opinion of Dr. Kinsbourne. Although he did not cite to medical literature to support this opinion, other decisions support a similar time period. *E.g., Murray v. Sec'y, HHS*, No. 99-545V, 2009 WL 3288300, at *26-28 (Fed. Cl. Spec. Mstr. Oct. 5, 2009) (finding a three to four day time period to be an appropriate temporal relationship between vaccination and injury).

Although not precisely applicable to the facts of this case, I note that brachial neuritis is a Table injury associated with tetanus-toxoid-containing vaccines. *See* § 14 *amended by* 42 C.F.R. § 100.3(a) (2011) (Vaccine Injury Table). Brachial neuritis may be caused through a mechanism similar to the theories advanced in this case. *See* 2012 IOM Report at 340 ("Autoantibodies, T cells, and complement activation may contribute to the symptoms of brachial neuritis," (in discussing influenza vaccine and brachial neuritis)). As set forth in the Vaccine Injury Table, onset occurring between 2-14 days for tetanus-toxoid-vaccine induced brachial neuritis constitutes the temporal relationship establishing the Table injury. Although this time frame may represent a policy as well as a scientific assessment, it does tend to indicate that onset of another autoimmune neurological injury between three and four days after vaccination is not, per se, unreasonable.

[41] *See Hanlon v. Sec'y, HHS*, 40 Fed. Cl. 625, 629-30 (1998).

Although Dr. Mateen did not address timing specifically in opining that HTR's ATM represented a vaccine injury, he was aware of the timing between vaccine and onset in this case. Pet. Ex. 54 at 1 (noting that HTR "received Gardasil, Menatra, and tetanus vaccinations within one week prior to her symptom onset").

7. Conclusions Regarding the *Althen* Factors.

In the absence of evidence refuting Dr. Kinsbourne's causation opinions, and in the presence of evidence supporting them, I find adequate evidence to conclude that ATM can be caused by tetanus vaccinations and that it was so caused in this case. I also find preponderant evidence that HTR's tetanus vaccination caused her to develop ATM within a medically appropriate time period.

VI. CONCLUSION.

Petitioners are entitled to compensation for HTR's condition. Having informally communicated my causation decision to the parties in a status conference on November 15, 2013, the parties have begun working on damages, and petitioners have retained a life care planner to assist in determining HTR's future needs. A specific damages order, if necessary, will follow the next status conference in this case.

IT IS SO ORDERED.

s/ Denise K. Vowell
Denise K. Vowell
Chief Special Master